

## Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium:

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## Periodontal Health and Gingival Diseases/Conditions.

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**Running head:** Classification and case definitions for periodontal health and gingival diseases and conditions.

**Key words:** periodontal disease, clinical health, dental plaque-induced gingivitis, non-dental plaque-induced gingival conditions, intact periodontium, stable periodontitis, reduced periodontium, disease stability, disease control, disease remission, modifying factors, predisposing factors, local risk factors, systemic risk factors, puberty, pregnancy, menstrual cycle, oral contraceptive, hyperglycemia, leukemia, smoking, restoration margins, hyposalivation, drug-induced gingival enlargement, symbiosis, dysbiosis, resolution of inflammation, biofilm, hereditary gingival fibromatosis, necrotizing periodontal diseases, *Neisseria gonorrhoea*, *Treponema pallidum*, *Mycobacterium tuberculosis*, Streptococcal gingivitis, Coxsackie virus, hand foot and mouth, herpangina, Herpes simplex, Varicella zoster, Molluscum contagiosum, squamous cell papilloma, condylomata acuminatum, verruca vulgaris, focal epithelial hyperplasia, Candidosis, Histoplasmosis, Aspergillosis, Coccidiomycosis, Paracoccidiomycosis, mucormycosis, geotricosis, blastomycosis, contact allergy, plasma cell gingivitis, erythema multiforme,

pemphigus vulgaris, pemphigoid, lichen planus, lupus erythematosus, orofacial granulomatosis, Crohn's disease, sarcoidosis, Melkersson-Rosenthal, allergic reaction, fibrous epulis, vascular epulis, pyogenic granuloma, calcifying fibroblastic granuloma, peripheral giant cell granuloma, leukoplakia, erythroplakia, squamous cell carcinoma, leukemia, Hodgkin's lymphoma, non-Hodgkins lymphoma, scurvy, frictional keratosis, toothbrush trauma, factitious injury, chemical trauma, thermal trauma, gingival pigmentation, melanoplakia, drug-induced pigmentation, smokers melanosis, amalgam tattoo.

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## ABSTRACT

Periodontal health is defined by absence of clinically detectable inflammation. There is a biological level of immune surveillance that is consistent with clinical gingival health and homeostasis. Clinical gingival health may be found in a periodontium that is intact, i.e. without clinical attachment loss or bone loss, and on a reduced periodontium in either a non-periodontitis patient (e.g. in patients with some forms of gingival recession or following crown lengthening surgery) or in a patient with a history of periodontitis who is currently periodontally stable. Clinical gingival health can be restored following treatment of gingivitis and periodontitis. However, the treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis and accordingly must be closely monitored.

Two broad categories of gingival diseases include non-dental plaque biofilm-induced gingival diseases and dental plaque-induced gingivitis. Non-plaque induced gingival diseases include a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal. Such lesions may be manifestations of a systemic condition or may be localized to the oral cavity. Dental plaque-induced gingivitis has a variety of clinical signs and symptoms, and both local predisposing factors and systemic modifying factors can affect its extent, severity and progression. Dental plaque-induced gingivitis may arise on an intact periodontium or on a reduced periodontium in either a non-periodontitis patient or in a currently stable “periodontitis patient” i.e. successfully treated, in whom clinical inflammation has been eliminated (or substantially reduced). A periodontitis patient with gingival inflammation remains a periodontitis patient (Figure 1), and comprehensive risk assessment and management are imperative to ensure early prevention and/or treatment of recurrent/progressive periodontitis.

Precision dental medicine defines a patient-centered approach to care and therefore creates differences in the way in which a “case” of gingival health or gingivitis is defined for clinical practice as opposed to epidemiologically in population prevalence surveys. Thus, case definitions of gingival health and gingivitis are presented for both purposes. While gingival health and gingivitis have many clinical features, case definitions are primarily predicated on presence or absence of bleeding on probing. Here we classify gingival health and gingival diseases/conditions, along with a summary table of diagnostic features for defining health and gingivitis in various clinical situations.

# **Periodontal health, gingival diseases and conditions on an intact and a reduced periodontium**

## **Workgroup 1: Periodontal Health & Gingival Diseases/Conditions**

“Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity”.<sup>1</sup> Based upon this definition from the World Health Organization (WHO), it follows that periodontal health should be defined as a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease. Based upon this overall framework of health, periodontal health should be predicated upon the absence of disease, as assessed clinically, associated with gingivitis, periodontitis, or other periodontal conditions, and may include patients who have had a history of successfully treated gingivitis or periodontitis, or other periodontal conditions, who have been and are able to maintain their dentition without signs of clinical gingival inflammation. Additionally, clinical periodontal health embraces physiological immune surveillance involving levels of biological and inflammatory markers compatible with homeostasis.<sup>2</sup> Periodontitis is a chronic inflammatory disease that currently can be successfully controlled and teeth retained for life. Periodontitis can remain stable (in remission) or enter periods of exacerbation. A stable periodontitis patient remains at higher risk for recurrent disease compared to a gingivitis patient or a healthy patient. Therefore, precision dental medicine requires ongoing, individual risk assessment as part of optimal patient management.

A definition of periodontal health and wellness is critical to establish ideal and acceptable therapeutic endpoints to periodontal therapies, to systematically assess the biological burden of periodontal inflammation to categorize gingival and periodontal disease prevalence in populations, and to evaluate individualized risk for future disease development. Periodontal health must be assessed and defined at both the patient and the site level to achieve these goals. Furthermore, definitions of periodontal health that are used to inform treatment decisions for individual patients may differ from those used in epidemiological studies.

### **Is there a level of gingival inflammation that is consistent with clinical periodontal health at a site level?**

There is a biological level of immune surveillance, manifesting as a predominantly neutrophilic infiltrate that is consistent with clinical gingival health.<sup>2</sup>

### **What is the spectrum of clinical periodontal health at a site level?**

*What is the biology of clinical gingival health?*

Clinical gingival health is generally associated with an inflammatory infiltrate and a host response consistent with homeostasis.

*On a site level, how do we classify clinical gingival health?*

- Clinical gingival health on an intact periodontium
- Clinical gingival health on a reduced periodontium
  - Stable periodontitis patient
  - Non-periodontitis patient (e.g. recession, crown lengthening)

*What are the clinical features of gingival health on an intact periodontium?*

Clinical gingival health on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss. Physiological bone levels range from 1.0 to 3.0 mm from the cemento-enamel junction.

*What are the clinical features of gingival health on a reduced periodontium?*

Clinical gingival health on a reduced periodontium is characterized by an absence of bleeding on probing, erythema, edema and patient symptoms in the presence of reduced clinical attachment and bone levels. However, it should be recognized that successfully treated and stable periodontitis patients remain at increased risk of recurrent progression of periodontitis. In non-periodontitis patients, there is no current evidence for increased risk of periodontitis.

*What are the clinical features of gingival health following treatment of gingivitis on an intact periodontium?*

Clinical gingival health following treatment of gingivitis on an intact periodontium is characterized by the absence of bleeding on probing, erythema and edema, patient symptoms, and attachment and bone loss.

*What are the clinical features of gingival health following successful treatment of periodontitis?*

Clinical gingival health following successful treatment of periodontitis is characterized by an absence of bleeding on probing, erythema, edema and patient symptoms in the presence of reduced clinical attachment and bone levels.

### **Case Definitions for Periodontal Health and Gingivitis:**

Based on available methods to assess gingival inflammation, a gingivitis case can be simply, objectively and accurately defined and graded using a bleeding on probing score (BoP%),<sup>3</sup> assessed as the proportion of bleeding sites (dichotomous yes/no evaluation) when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled (~0.2 N) force to the apical end of the sulcus at 6 sites (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, disto-lingual) on all teeth present. Limitations of these clinical criteria arise from a lack of standardized periodontal probes (e.g. probe dimensions, taper), examiner variability (probe pressure, angle), patient related factors (biotype, medications, etc.) and smoking.

**In all references to an "intact periodontium" within this consensus, an absence of detectable attachment and/or bone loss is implicit.**

**How do we define a case of gingival health on an intact and a reduced periodontium for epidemiological purposes?**

For an intact periodontium **and a reduced and stable periodontium**, gingival health is defined as <10% bleeding sites<sup>4,5</sup> with probing depths ≤ 3mm.

## How do we define a case of gingival health on an intact and a reduced periodontium for clinical practice?

Due to limitations in, and a lack of uptake of, standardized ISO probes and techniques leading to inherent measurement variability in the parameters of gingival health, a patient with periodontal health may exhibit one or two sites with some evidence of clinical gingival inflammation. Moreover, localized mild and delayed bleeding to probe at isolated sites is ubiquitous, but may fall within the spectrum of "clinical health".

In clinical practice, a case of gingival health on an intact periodontium would be a patient with no signs of gingivitis as defined above.

In clinical practice, the goal of periodontal treatment on a reduced periodontium is a patient with no signs of gingivitis as defined above. A case of gingival health on a reduced periodontium in a stable periodontitis patient must be distinguished from a case of periodontal health in a reduced periodontium in a non-periodontitis patient (recession, crown lengthening), because there is a difference in risk for periodontal disease progression.

Following treatment of periodontitis, periodontitis patients may not attain a status of complete gingival health based on the above definition. However, evidence has demonstrated that a patient may achieve periodontal stability. Periodontal stability is characterized by successful treatment through control of local and systemic risk factors, resulting in minimal (<10% of sites<sup>5</sup>) BOP, no probing depths of 4mm or greater that bleed on probing, optimal improvement in other clinical parameters and lack of progressive periodontal destruction. The treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis and accordingly must be closely monitored. Figure 1 summarizes the various scenarios that may arise following the transition from health, to gingivitis and ultimately periodontitis.

## How do we define gingivitis at a site level (biological & clinical)?

Defining inflammation at a site level is quite distinct from defining a case of gingivitis. A universal case definition is essential to facilitate population surveillance, for clinicians setting therapeutic targets, and to enable assessment of the efficacy of prevention and/or treatment regimes.

There are broadly two categories of gingival disease:

- Dental plaque biofilm-induced gingivitis
- Non-dental plaque-induced gingival diseases

Dental plaque biofilm-induced gingivitis is defined at the site level as "an inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune-inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone). Such inflammation remains confined to the gingiva and does not extend beyond the mucogingival junction and is reversible by reducing levels of dental plaque at and apical to the gingival margin".

Depending on whether dental plaque biofilm-induced gingival inflammation occurs on an intact or reduced periodontium, or in a patient diagnosed with periodontitis, gingivitis can be further classified as:

- Gingivitis on an intact periodontium
- Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g. recession, crown lengthening)
- **Gingival inflammation** on a reduced periodontium in a successfully treated periodontitis patient (Note that recurrent periodontitis cannot be ruled out in this case)

Since the 1999 classification, there have been advances in knowledge of the microbiome and the gingival transcriptome. Gingivitis is a non-specific inflammatory condition and is therefore a consequence of sustained plaque biofilm accumulation at and apical to the gingival margin<sup>7</sup>. Longitudinal studies have demonstrated that sites that do not progress to attachment loss are characterized by less gingival inflammation over time, whereas those sites that do progress have persistently greater levels of gingival inflammation<sup>8-13, 14</sup>. Therefore, gingivitis is a major risk factor, and a necessary pre-requisite, for periodontitis. The management of gingivitis is thus a primary prevention strategy for periodontitis.

Periodontitis patients who are currently stable but develop **gingival inflammation** at specific sites should remain on periodontal maintenance and should be closely monitored during periodontal maintenance for any reactivation of periodontitis. Such patients may not be managed in the same way as non-periodontitis patients with gingivitis.

### **What are the determinants of the rate of development of gingivitis, its severity and extent?**

The threshold of plaque accumulation necessary to induce gingival inflammation and impact upon its rate of progression at specific sites or at a whole mouth level varies between individuals according to both local risk factors, known as predisposing factors, and systemic risk factors, referred to as modifying factors, respectively.

#### *1) Local risk factors (predisposing factors)*

Local risk factors for gingivitis are those that encourage plaque accumulation at a specific site by either inhibiting its removal during daily oral hygiene practices, and/or creating a biological niche that encourages increased plaque accumulation<sup>7</sup>. These include:

- a) Dental plaque biofilm retention factors (including certain tooth anatomical factors) – facilitate plaque accumulation at and apical to the gingival margin, enabling biofilm adherence and maturation and increasing the difficulty of mechanical plaque removal. Several clinical studies providing a moderate level of evidence have demonstrated that sub-gingival restoration margins may be detrimental to gingival health<sup>15, 16</sup>.
- b) Oral dryness is a clinical condition often associated with symptoms of xerostomia. Oral dryness manifesting as a lack of salivary flow, availability, or changes in quality of saliva, leading to reduced cleansing of tooth surfaces is associated with reduced dental plaque biofilm removal and enhanced gingival inflammation. Common causes include medications that have anti-



parasympathetic action, Sjögrens syndrome when the salivary acini are replaced by fibrosis following autoimmune destruction, and mouth breathing in people who may have enhanced gingival display and/or an incompetent lip seal<sup>17</sup>.

## 2) Systemic risk factors (modifying factors)

Systemic risk or modifying factors are those characteristics present in an individual, which negatively influence the immune-inflammatory response to a given dental plaque biofilm burden, resulting in exaggerated or “hyper” inflammation. Examples include:

- a) Smoking – is one of the major lifestyle/behavioral risk factors for periodontitis, but which also has profound effects upon the gingival tissues. Systemic circulatory uptake of components of cigarette smoke as well as local uptake are reported to induce microvascular vasoconstriction and fibrosis. This can mask clinical signs of gingivitis, such as bleeding on probing, despite a significant underlying pathological inflammatory cell infiltrate<sup>18</sup>.
- b) Metabolic factors – hyperglycemia in people with or without diabetes. Excess glucose is toxic and directly induces mitochondrial stress and an enhanced respiratory burst in inflammatory cells that may activate various pro-inflammatory mediator cascades. Formation of advanced glycation end-products (AGEs) may also result in AGE binding to its cell surface receptor (RAGE), which activates pro-inflammatory signaling cascades and downstream pro-inflammatory events.
- c) Nutritional factors - Severe vitamin C deficiency, or scurvy, results in compromised antioxidant micronutrient defenses to oxidative stress and also negatively impacts collagen synthesis, resulting in weakened capillary blood vessel walls and a consequent propensity to enhanced gingival bleeding<sup>20</sup>.
- d) Pharmacological agents (prescription, non-prescription and recreational agents) – can act via diverse mechanisms to increase susceptibility to gingivitis. This may include drugs that reduce salivary flow, drugs that impact endocrine function (see below), and drugs that may induce gingival enlargement and pseudo-pocketing.
- e) Elevations in sex steroid hormones – at puberty, during pregnancy or following medication with first generation oral contraceptives may modify the gingival inflammatory response. Complex biological reactions within the gingival tissues result from such elevated sex steroid levels and generate more than expected inflammation, in response to relatively small levels of plaque. However, modern oral contraceptive dosages have been reduced and there is little evidence for exaggerated gingival inflammatory responses to plaque with such drugs<sup>21</sup>.
- f) Hematological conditions – particular blood malignancies such as leukemia or pre-malignant conditions such as myelodysplasia are associated with signs of excess gingival inflammation in the absence of excessive plaque biofilm accumulation. Signs include swollen, purple or occasionally pale gingiva due to leukemic cell infiltration, gingival bleeding that is inconsistent with levels of dental plaque biofilm accumulation, due to thrombocytopenia and/or clotting factor deficiencies<sup>22</sup>.

### **What are the diagnostic criteria for a gingivitis case?**

Given the “spectrum” of presentation of gingival health and gingival inflammation in terms of severity and extent of gingival involvement, it is important to define the features of a universally accepted case of gingivitis.

Current epidemiological data on the prevalence of gingivitis suffer from the lack of a universally adopted case definition and vary as widely as 6% to 94%, due to the use of indices that measure gingival inflammation at individual sites rather than considering the patient’s mouth as a whole. Therefore, mild localized clinical inflammation is reported to affect almost 95% of the population, a figure that would incorrectly suggest gingivitis as being a variation of “normality” and thus consistent with the spectrum of “clinical health” rather than being a disease. By contrast, the more extensive the manifestation of disease employed in a case definition, the lower the reported prevalence. A universally agreed case definition should be based upon a pragmatic appraisal of the evidence base derived from longitudinal observation and intervention studies.

#### *Clinical, radiological, biological signs and symptoms*

1. Gingivitis is a clinical diagnosis. While emerging technologies are starting to shed light on the microbiological, molecular and pathophysiological characteristics of gingivitis, definitive knowledge is not sufficient to supersede current clinical parameters<sup>7</sup>.
2. The clinical signs of inflammation are erythema, edema, pain (soreness), heat and loss of function.
3. These may manifest clinically in gingivitis as
  - a. Swelling, seen as loss of knife edged gingival margin and blunting of papillae
  - b. Bleeding on gentle probing
  - c. Redness
  - d. Discomfort on gentle probing
4. The symptoms a patient may report include
  - a. Bleeding gums (metallic/altered taste)
  - b. Pain (soreness)
  - c. Halitosis
  - d. Difficulty eating
  - e. Appearance (swollen red gums)
  - f. Reduced oral health related quality of life
5. Radiographs cannot be used to diagnose gingivitis.

### **Should we classify dental plaque biofilm-induced gingivitis?**

There is utility in defining the severity of gingivitis as a patient communication tool, but there are no objective clinical criteria for defining severity. Thus, in this context alone, the extent of gingivitis can be used to communicate “mild, moderate and severe” gingivitis. Moreover, emerging evidence suggests that the contained gingivitis lesion may have systemic inflammatory consequences<sup>23,24</sup>.

There is no robust evidence to clearly differentiate mild, moderate and severe gingivitis and definitions remain a matter of professional opinion. Methods of defining gingivitis may include:

1. Defining percentages (e.g. mild = <10%, moderate = 10-30%, severe = >30% sites)
2. Grading (e.g. grade 1-5 in 20% quintiles for % sites bleeding on probing)

### **How do we define a case of dental plaque-induced gingivitis on an intact and a reduced periodontium for epidemiological purposes?**

For epidemiological purposes, gingivitis on an intact periodontium and gingivitis on a reduced periodontium in a patient without a history of periodontitis, is defined as  $\geq 10\%$  bleeding sites<sup>4,5</sup> with probing depths  $\leq 3\text{mm}$ . Localized gingivitis is defined as 10%-30% bleeding sites; generalized gingivitis is defined as  $>30\%$  bleeding sites.

For epidemiological purposes alone, a periodontitis case cannot simultaneously be defined as a gingivitis case. Therefore, a patient with a history of periodontitis, with gingival inflammation is still a periodontitis case.

### **How do we classify a patient with dental plaque-induced gingivitis on an intact and a reduced periodontium for clinical practice?**

In clinical practice, a case of gingivitis on an intact periodontium, or a reduced periodontium in a patient without a history of periodontitis, would be a patient with signs of gingival inflammation as defined above (Table 1).

In clinical practice, periodontitis patients, if successfully treated can achieve a reduced and stable periodontium where probing pocket depths are  $\leq 4\text{mm}$ <sup>27</sup> and there is an absence of clinical inflammation (bleeding on probing). **Gingival inflammation** may arise at specific sites, and where probing depths are  $\leq 3\text{mm}$  is termed **gingival inflammation** in a stable periodontitis patient. However, such patients remain at high risk of recurrent periodontitis and require close monitoring as such sites are at high risk of reverting to periodontitis (Table 1).

### **How do we classify non-dental plaque-induced gingival conditions?**

Although oral health and systemic health are frequently considered as separate entities, both are strongly interrelated. There are numerous examples of how oral diseases may impact systemic health and how the oral cavity may be a window to general health. Consequently, it is crucial for all health care providers to understand these interrelationships, inform patients of such conditions, and make appropriate referrals.

Non dental-plaque induced gingival conditions encompass a variety of conditions that are not caused by plaque and usually do not resolve following plaque removal. Such lesions may be manifestations of a systemic condition or may be localized to the oral cavity<sup>25</sup>. Although these lesions are not caused by the dental plaque biofilm, the severity of the clinical manifestations often depends on plaque accumulation and subsequent gingival inflammation<sup>26</sup>.

The proposed classification considers those conditions listed in Table 2.

### **Which non-dental-plaque-induced gingival conditions may have associated systemic involvement and how does that impact upon patient-centered care pathways?**

In recent years, the traditional treatment model in which the patient was a passive receiver of care is changing toward patient-centered care in Precision Dental Medicine (PDM). In PDM, an individual's specific health needs and desired health

outcomes are the driving force behind all health care decisions and quality measurements. One of the elements in PDM is that care is collaborative, coordinated and accessible. The right care is provided at the right time and the right place. Considering that the conditions marked with an asterisk (Table 2) have associated systemic involvement or are oral manifestations of systemic conditions, other health care providers may be involved in diagnosis and treatment.

## **Future Research Needs**

Regarding classification and diagnosis of periodontal health and gingival diseases/conditions, future research is needed on the:

- development and validation of non-invasive diagnostic tools (e.g., saliva-based diagnostics), especially as they relate to detection of gingival inflammation;
- identification of the characteristics (e.g., genetic factors) that distinguish persons who are resistant to the development of dental plaque biofilm-induced or non-dental plaque biofilm-induced gingival diseases from those who are susceptible;
- expansion of our limited knowledge of the determinants that affect the reliability of currently available diagnostic tools (e.g., effects of probe design on bleeding on probing responses);
- characterization of the possible differences (e.g., molecular determinants) between gingivitis on an intact periodontium and other forms of gingival inflammatory disease.

Regarding the current primary periodontal diagnostic tool, the graduated periodontal measuring probe, the following are recommendations for an ISO periodontal probe:

The reliability and reproducibility of any case definition for health, gingival or periodontal conditions relies upon standardization of probing protocols, which is only possible with the implementation of an ISO probe. The current International standard for organization (ISO) for periodontal probes is - ISO 21672, but requires updating in order to define the features of a global standard periodontal probe. These characteristics are:

1. Tip diameter 0.5mm
2. Cylindrical tine structure
3. Constant force limiter of 0.2N
4. 15mm scale with precise individual or banded millimeter markings
5. A taper of 1.75°

## References

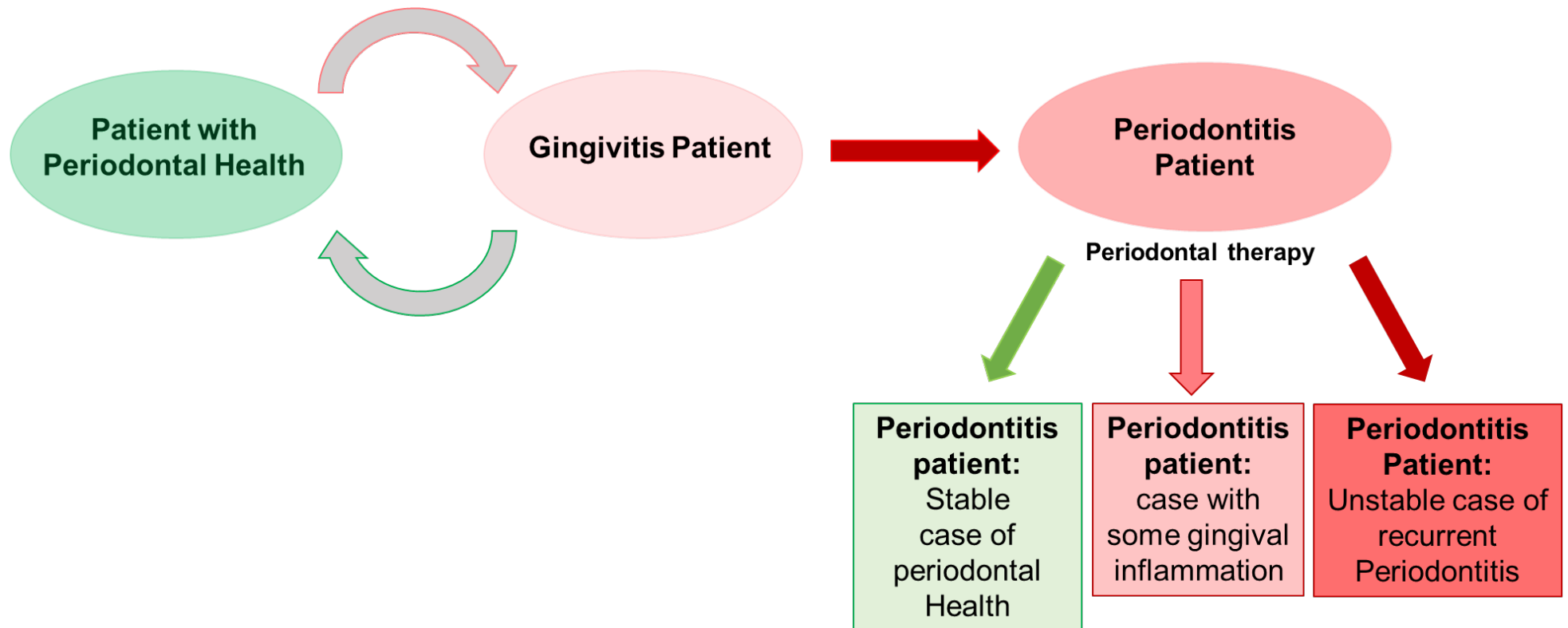
1. World Health Organization. Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference. Official Records of the World Health Organization 1948; 19456 No. 2:1.
2. Lang NP, Bartold PM. Periodontal Health. 2018. **ADD CITATION FOR THIS PAPER FROM THIS JOURNAL**
3. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J* 1975;25:229-235.
4. Trombelli L, Farina R, Silva CO, Tatakis DN. Plaque-induced gingivitis. Case definition and diagnostic considerations. 2017. **ADD CITATION FOR THIS PAPER FROM THIS JOURNAL**
5. Ramseier C, Mirra D, Schütz C, Sculean A, Lang NP, Walter C, Salvi GE. Bleeding on probing as it relates to smoking status in patients enrolled in supportive periodontal therapy for at least 5 years. *J Clin Periodontol* 2015;42:150-159.
6. Matuliene G, Pjetursson BE, Salvi GE, Schmidlin K, Bragger U, Zwahlen M, Lang NP. Influence of residual pockets on progression of periodontitis and tooth loss: Results after 11 years of maintenance. *J Clin Periodontol* 2008;35:685-695.
7. Murakami S, Mealey B, Mariotti A, Chapple ILC. Dental plaque induced gingival conditions. 2018. **ADD CITATION FOR THIS PAPER FROM THIS JOURNAL**
8. Loe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. *J Clin Periodontol* 1986;13:431-445.
9. Ismail AI, Morrison EC, Burt BA, Caffesse RG, Kavanagh MT. Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959-87. *J Dent Res* 1990;69:430-435.
10. Clerehugh V, Worthington HV, Lennon MA, Chandler R. Site progression of loss of attachment over 5 years in 14- to 19-year-old adolescents. *J Clin Periodontol* 1995;22:15-21.
11. Albandar JM, Kingman A, Brown LJ, Loe H. Gingival inflammation and subgingival calculus as determinants of disease progression in early-onset periodontitis. *J Clin Periodontol* 1998;25:231-237.
12. Schätzle M, Loe H, Bürgin W, Anerud A, Boysen H, Lang NP. Clinical course of chronic periodontitis. I. Role of gingivitis. *J Clin Periodontol* 2003;30:887-901. Erratum in: *J Clin Periodontol* 2004;31:813.
13. Ramseier CA, Ånerud Å, Dulac M, Lulic M, Cullinan MP, Seymour GJ, Faddy MJ, Bürgin W, Schätzle M, Lang NP. Natural history of periodontitis: Disease

progression and tooth loss over 40 years. *J Clin Periodontol* 2017 (epub ahead of print).

14. Tanner ACR, Kent R Jr., Kanasi E, Lu SC, Paster BJ, Sonis ST, Murray LA, Van Dyke TE. Clinical characteristics and microbiota of progressing slight chronic periodontitis in adults. *J Clin Periodontol* 2007;34:917-930.
15. Lang NP, Kiel RA, Anderhalden K. Clinical and microbiological effects of subgingival restorations with overhanging or clinically perfect margins. *J Clin Periodontol* 1983;10:563-578.
16. Schätzle M, Land NP, Anerud A, Boysen H, Bürgin W, Loe H. The influence of margins of restorations of the periodontal tissues over 26 years. *J Clin Periodontol* 2001;28:57-64.
17. Mizutani S, Ekuni D, Tomofuji T, Azuma T, Kataoka K, Yamane M, Iwasaki Y, Morita M. Relationship between xerostomia and gingival condition in young adults. *J Periodont Res* 2015;50:74-79.
18. Warnakulasuriya S, Dietrich T, Bornstein MM, Casals Peidró E, Preshaw PM, Walter C, Wennström JL, Bergström J. Oral health risks of tobacco use and effects of cessation. *Int Dent J* 2010;60:7-30.
19. Chapple ILC, Genco R. Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Disease. *J Clin Periodontol* 2013;40(S14):106-12.
20. Van der Velden U, Kuzmanova D, Chapple ILC. Micronutritional approached to periodontal therapy. *J Clin Periodontol* 2011;38(s11):142-158.
21. Trombelli L, Farina R. A review of factors influencing the incidence and severity of plaque-induced gingivitis. *Minerva Stomatol* 2013;62:207-234.
22. Lynch MA, Ship II. Initial oral manifestations of leukemia. *JADA* 1967;75:932-940.
23. Wohlfeil M, Wehner J, Schacher B, Oremek GM, Sauer-Eppel H, Eickholz P. Degree of gingivitis correlates to systemic inflammation parameters. *Clin Chim Acta* 2009;401:105-109.
24. Eberhard J, Grote K, Luchtefeld M, Heuer W, Schuett H, et al. Experimental gingivitis induced systemic inflammatory markers in young healthy individuals: a single-subject interventional study. *PLoS One* 2013;8:e55265.
25. Holmstrup P, Plemons J, Meyle J. Non-plaque induced gingival diseases. 2018. **ADD CITATION FOR THIS PAPER FROM THIS JOURNAL.**

26. Stone SJ, Heasman PA, Staines KS, McCracken GI. The impact of structured plaque control for patients with gingival manifestations of oral lichen planus: a randomized controlled study. *J Clin Periodontol* 2015;42:356–362.
27. Wennström JL, Tomasi C, Bertelle A, Dellasega E. Full-mouth ultrasonic debridement versus quadrant scaling and root planing as an initial approach in the treatment of chronic periodontitis. *J Clin Periodontol* 2005;32:851–859.

Figure 1



**Figure Legend:**

The transition from periodontal health to gingivitis is reversible following treatment that resolves gingival inflammation. The transition to periodontitis results in attachment loss which, at the present time is irreversible. More importantly, it signposts patients who are at lifelong high risk of recurrent periodontitis. Optimal periodontal therapy can restore gingival health on a reduced periodontium, or may result in mild marginal gingival inflammation at shallow probing pocket depths ( $\leq 3\text{mm}$ ). However, a history of periodontitis places patients at high risk of recurrent periodontitis and such patients require careful site-specific monitoring during periodontal maintenance programs.



**Table 1**

**Diagnostic look up table for gingival health or dental plaque-induced gingivitis in clinical practice.**

<b>Intact periodontium</b>	<b>Health</b>	<b>Gingivitis</b>
<i>Probing attachment loss</i>	No	No
<i>Probing pocket depths (assuming no pseudo pockets)</i>	≤3mm	≤3mm
<i>Bleeding on probing</i>	<10%	Yes (≥10%)
<i>Radiological bone loss</i>	No	No
<b>Reduced periodontium Non periodontitis patient</b>	<b>Health</b>	<b>Gingivitis</b>
<i>Probing attachment loss</i>	Yes	Yes
<i>Probing pocket depths (all sites &amp; assuming no pseudo pockets)</i>	≤3mm	≤3mm
<i>Bleeding on probing</i>	<10%	Yes (≥10%)
<i>Radiological bone loss</i>	Possible	Possible
<p>NB: In conditions where there is treatment but not cure, e.g. rheumatoid arthritis, periodontitis, the post-treatment parameters that define stability/health or gingivitis may differ from the parameters for health/gingivitis in a non-periodontitis patient. The threshold for “clinical health” in a treated and stable periodontitis patient is therefore set at ≤4mm.</p>		
<b>Successfully treated stable periodontitis patient</b>	<b>Health</b>	<b>Gingivitis in a patient with a history of periodontitis</b>
<i>Probing attachment loss</i>	Yes	Yes
<i>Probing pocket depths (all sites &amp; assuming no pseudo pockets)</i>	≤4mm (no site ≥ 4mm with BoP)**	≤3mm
<i>Bleeding on probing</i>	<10%	Yes (≥10%)
<i>Radiological bone loss</i>	Yes	Yes
<p><b>NB: A successfully treated periodontitis patient in whom sites of gingival bleeding appear, remains at high risk of disease recurrence at those sites and of progressive attachment loss. Therefore, gingivitis is defined as bleeding at a shallow site of ≤3mm rather than ≤4mm, as is the case in gingival health. Where the probing depth is 4mm or higher with bleeding, this is no longer a “closed pocket”<sup>21</sup></b></p>		

**\* Assumes a light probing pressure of 0.2-0.25N**

**\*\* There was a rational minority view expressed that the threshold for defining a clinical case of health in a successfully treated periodontitis patient should be set at ≤3mm with no BoP to acknowledge the elevated risk of recurrent disease. However, the counter and majority view was that the ≤3mm threshold is rarely achieved at 100% of treated sites and could lead to over-treatment, since any non-bleeding site >3mm would not be classified as “health” and thus open to further invasive treatment, rather than monitoring and supportive care. The threshold was therefore set at ≤4mm acknowledging that post-treatment clinical phenotypes need to be considered differently to pre-treatment phenotypes.**

**Table 2**

**Classification of gingival health and gingival diseases/conditions**

**1. Periodontal Health<sup>2</sup>**

- A. Clinical health on an intact periodontium
- B. Clinical gingival health on a reduced periodontium
  - i) Stable periodontitis patient
  - ii) Non-periodontitis patient

**2. Gingivitis – dental plaque-induced: *Intact periodontium; Reduced periodontium in non-periodontitis patient; Reduced periodontium in successfully treated periodontitis patient.*<sup>7</sup>**

- A. Associated with biofilm alone
- B. Mediated by systemic or local risk factors
  - i) Systemic risk factors (Modifying factors)
    - a) Smoking
    - b) Hyperglycemia
    - c) Nutritional Factors
    - d) Pharmacological agents (prescription, non-prescription and recreational)
    - e) Sex steroid hormones
      - Puberty
      - Menstrual cycle
      - Pregnancy
      - Oral contraceptives
    - f) Hematological conditions
  - ii) Local risk factors (Predisposing factors)
    - a) Dental plaque biofilm retention factors (e.g., prominent restoration margins)
    - b) Oral dryness
- C. Drug-influenced gingival enlargement

**3. Gingival diseases - Non-dental plaque-induced<sup>25</sup>**

- A. Genetic/Developmental Disorders
  - i) Hereditary gingival fibromatosis \*
- B. Specific Infections
  - i) Bacterial origin
    - a) Neisseria gonorrhea \*
    - b) Treponema pallidum \*
    - c) Mycobacterium tuberculosis \*
    - d) Streptococcal gingivitis
  - ii) Viral origin
    - a) Coxsackie virus (Hand foot and mouth disease) \*
    - b) Herpes simplex I & II (primary or recurrent) \*
    - c) Varicella zoster (Chicken pox & shingles – V nerve) \*
    - d) Molluscum contagiosum \*
    - e) Human papilloma virus (squamous cell papilloma; Condyloma acuminatum; Verruca vulgaris; focal epithelial hyperplasia)

- iii) Fungal origin
  - a) Candidosis
  - f) Other mycoses (e.g. Histoplasmosis; Aspergillosis)

#### C. Inflammatory and immune conditions

- i) Hypersensitivity reactions
  - a) Contact allergy \*
  - b) Plasma cell gingivitis \*
  - c) Erythema multiforme \*
- ii) Autoimmune diseases of skin and mucous membranes
  - a) Pemphigus vulgaris \*
  - b) Pemphigoid \*
  - c) Lichen planus \*
  - d) Lupus erythematosus \*
    - Systemic lupus erythematosus
    - Discoid lupus erythematosus
- iii) Granulomatous inflammatory lesions (oro-facial granulomatoses)
  - e) Crohn's disease \*
  - f) Sarcoidosis \*

#### D. Reactive processes

- i) Epulides
  - a) Fibrous epulis
  - b) Calcifying fibroblastic granuloma
  - c) Vascular epulis (pyogenic granuloma)
  - d) Peripheral giant cell granuloma \*

#### E. Neoplasms

- i) Premalignancy
  - a) Leukoplakia
  - b) Erythroplakia
- ii) Malignancy
  - a) Squamous cell carcinoma \*
  - b) Leukemic cell infiltration \*
  - c) Lymphoma \*
    - Hodgkins
    - Non-Hodgkins

#### F. Endocrine, nutritional & metabolic diseases

- i) Vitamin deficiencies \*
  - a) Vitamin C deficiency (scurvy)

#### G. Traumatic lesions

- i) Physical/mechanical trauma
  - a) Frictional keratosis
  - b) Mechanically induced gingival ulceration
  - c) Factitious injury (self-harm)
- ii) Chemical (toxic) burn
- iii) Thermal insults
  - a) Burns to gingiva

#### H. Gingival pigmentation

- i) Melanoplakia \*
- ii) Smokers melanosis

- iii) Drug-induced pigmentation (anti-malarials, minocycline)
- iv) Amalgam tattoo

\* Conditions marked with an asterisk have associated systemic involvement or are oral manifestations of systemic conditions, therefore other health care providers may be involved in diagnosis and treatment.